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PESTS NOT KNOWN TO OCCUR IN THE UNITED STATES OR OF LIMITED DISTRIBUTION NO. 92: Synchytrium endobioticum

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Disease POTATO WART

Pathogen Synchytrium endobioticum (Schilberszky) Percival

Other Names Chrysophlyctis endobiotica Schilberszky
Synchytrium solani Massee

Class: Order Chytridiomycetes: Chytridiales

Economic Importance

Potato wart can cause severe losses. Susceptible potatoes in heavily infested soil can yield less than the weight of seed potatoes planted (Webster 1980), rarely producing more than 50 percent of a normal crop (Hartman and McCubbin 1924). Although planting resistant cultivars reduces crop losses to a minimum, this gain has been countered by the evolution of virulent pathotypes (races) that infect previously resistant cultivars (Bojňanský 1984). Because soil in infested fields is contaminated by the persistent, almost indestructible spores and movement of any material from such fields readily spreads the spores, additional economic losses may result. Sales are reduced because movement of apparently clean potatoes or nonhost crops from such soil must be confined to the infested area. No exports can be made because most countries prohibit imports from infested areas, due to the risk of introducing the pathogen or one of its more virulent pathotypes. Sales within the country or abroad of such material from the infested area are thus restricted for many years.

In Europe, the economic importance is currently most serious in Poland, Romania, the Soviet Union, and Switzerland (European and Mediterranean Plant Protection Organization 1980). Elsewhere, the disease is significant in Newfoundland, the Peruvian Andes of Peru and Bolivia, and parts of Nepal (International Potato Center 1979).

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Synchytrium endobioticum distribution map (Prepared by Technical Information Systems Staff, PPQ, APHIS, USDA).

General Distribution

Infestations occur in small areas (European and Mediterranean Plant Protection Organization 1974). The following countries were listed by the Commonwealth Mycological Institute (1983) unless otherwise cited: Austria, Belgium, Bhutan, Bolivia, Canada (Labrador and Newfoundland (Agriculture Canada 1985)), Chile, China (central and southern), Czechoslovakia, Denmark, East Germany, Ecuador, Falkland Islands, Faroe Islands, Finland, France, India (Darjeeling, Sikkim, West Bengal), Ireland, Italy, Luxembourg, Mexico (only on wild Solanum spp.), Nepal, Netherlands, New Zealand (southern South Island, under eradication), Norway, Peru, Poland, Romania, South Africa (Natal, Orange Free State, Transvaal), Soviet Union (Byelorussian S.S.R., Estonian S.S.R., Latvian S.S.R., Lithuanian S.S.R., Russian S.F.S.R., Ukrainian S.S.R. (Hampson 1979b)), Sweden, Switzerland, United Kingdom, Uruguay, West Germany, and Yugoslavia (Crna Gora).

This fungus has demonstrated its ability to establish itself in the United States, as shown from its discovery in 1918 in Pennsylvania and later finds in Maryland and West Virginia. Subsequently, eradication was successfully completed with the last detected infestation in Pennsylvania in the 1950's, Maryland in 1951 (L. O. Weaver, letter to W. Gimpel, Maryland Department of Agriculture, October 22, 1986), and West Virginia in 1969 (Brooks et al. 1974).

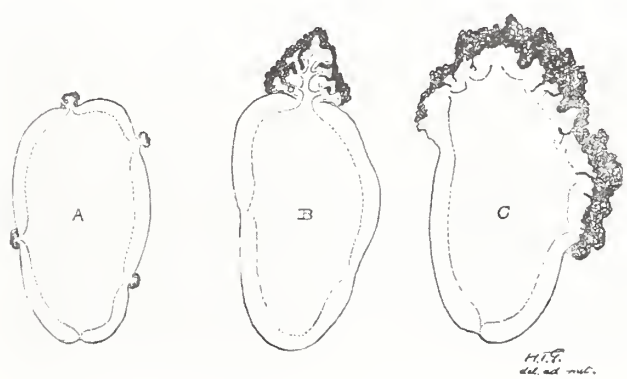
Hosts

Hosts belong to the Solanaceae. Naturally infected hosts include Solanum tuberosum (potato) (Walker 1983), the wild S. stoloniferum and S. vallis-mexici (Niederhauser 1953), and Lycopersicon lycopersicum (tomato) (Weiss and Brierley 1928, Weiss and Orton 1923).

Characters

WARTY OUTGROWTHS OR GALLS (Fig. 1) - Pea-sized or larger, developing at stem base, on tubers, and occasionally on aerial shoots of host; galls usually aggregated and confluent, forming large warts, dark brown to green depending on plant part infected, becoming black at maturity and later decaying to release resting spores. Tubers may be disfigured or completely replaced by galls. Roots are not known to be attacked.

(Fig. 1)



Synchytrium endobioticum warts on potato tuber, sectional views. A. Four eyes attacked. B. Branched wart from upper eye. C. Excrescence covering half of tuber (From Gussnow 1909).

RESTING SPORES - Deeply embedded in host tissue due to repeated division of infected cell; filling host cell almost completely; spherical to ovoid, 35-80 µm diameter, aseptate, golden-brown, thick-walled with outer wall furrowed, ridged (Fig. 2A),

(Fig. 2)

Synchytrium endobioticum resting spores. A. View of surface ridges. B. Germinating spore showing extruded vesicle (A from Hampson 1986; B, courtesy M. C. Hampson, Agriculture Canada).

irregularly thickened; each germinating spore gives rise to a thin-walled, hyaline vesicle (Fig. 2B) from which forms a hyaline sporangium. Zoospores develop and are eventually discharged.

ZOOSPORES - Spherical to elongate, 1.5-3.0 µm diameter, containing a large, eccentric, hyaline, refractive globule, posteriorly uniflagellate, flagellum of whiplash-type, 17 µm long; zoospores function as asexual infective agents which give rise to prosori or function as gametes which fuse to form biflagellate zygotes infecting the host and develop into resting spores.

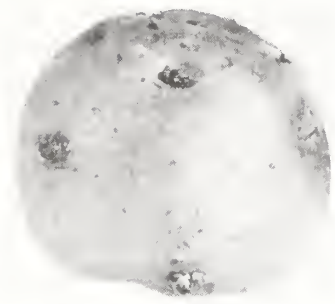
PROSORI - Arising from zoospores penetrating the host surface, 1-4 in an infected epidermal cell; each prosorus surrounded by enlarged host cells forming a rosette; occurring at host surface when mature due to host cell division and enlargement below the infected area; prosorus aseptate, pale golden-brown, thick-walled, smooth, 40-50 µm diameter, spherical to ovoid; contents emerging through a pore in outer prosorus wall to form an ovoid, flattened or almost spherical sorus of sporangia.

SPORANGIA - Aseptate, pale golden-brown, thinwalled, smooth, 25-38 x 62-87 µm, polyhedral, ovoid or almost spherical, 1-9 sporangia per sorus, germinating to release spores which function either as zoospores or gametes.

Characteristic Damage

The number of plants infected and the severity of the warts (Figs. 3-5) vary with the potato cultivar, pathotype, weather, soil moisture, inoculum concentration (Glynne 1925), and other factors. Aboveground symptoms are uncommon. Host growth may be retarded, or leaves may be darker green and slightly larger

(Figs. 3-5)



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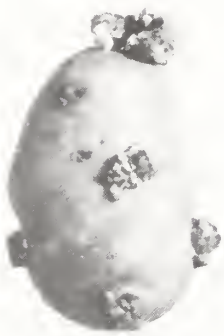

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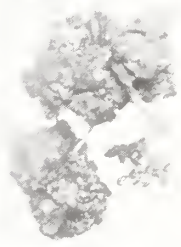

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Range of warty growths induced by Synchytrium endobioticum on potato. 3. Mild symptoms. 4. Moderate. 5. Severely diseased plant with all tubers affected, six covered by warty growths (3-4 from Weiss and Orton 1923; 5 from Gussnow 1909).

than normal. Small warts sometimes appear on leaves in contact with soil or at buds on stem bases. Below ground, warts often develop on stem bases, stolon tips, and tuber eyes, but not on roots (European and Mediterranean Plant Protection Organization 1980, Ireland Department of Agriculture and Fisheries 1968).

Warts are white at first, resembling small cauliflower heads, becoming green if exposed to light, then darkening to black. Wart size ranges from pinheads to growths larger than the tuber (Hampson and Proudfoot 1974, Ireland Department of Agriculture and Fisheries 1968). Warts at an advanced stage reduce the tuber to a brownish black, soft mass with a rotten odor. This mass readily falls apart during harvest (Spaulding and Field 1912).

In dry seasons or on immune cultivars, the tiny warts (Fig. 4) may be overlooked on harvested tubers. Such warts may be the same color as the tuber's skin (European and Mediterranean

Plant Protection Organization 1980). The disease may continue in storage, especially under the high humidity used to store potatoes for consumption (Noble and Glynne 1970). Susceptible potatoes completely rot in storage in 2-3 weeks (Hartman and McCubbin 1924).

Detection Notes

This pathogen is transported into new areas in infected potato tubers. Dispersal also occurs through movement of any materials contaminated by the long-lived, soilborne resting spores. Contaminated materials can include healthy tubers, nonhost planting material, soil, manure, containers, tools, machinery, footwear, and animals (Hampson 1981, Hartman and McCubbin 1924, Weiss and Brierley 1928). The risk of introduction is high because this fungus cannot be detected on contaminated seed potatoes or in slightly infected potatoes (Noble and Glynne 1970, Spaulding and Field 1912). With the fungus remaining dormant under the low-humidity storage used for seed potatoes, the pathogen can remain undetected from planting until after harvest (Noble and Glynne 1970). Many countries, thus, have strict quarantines in effect.

To prevent the introduction of the potato wart pathogen into the United States, Title 7 of the Code of Federal Regulations, Part 321 regulates the entry of potatoes and its Solanum relatives that produce tubers. Tubers may enter from countries certified free of Synchytrium endobioticum and other pests if these approved countries prohibit the entry of potatoes from other countries infested with injurious potato diseases. These certified tubers from approved countries may enter the United States under permit for any purpose, subject to inspection. Parts 319.69, 320, and 330.300 regulate the entry of soil.

In surveys,

1. Examine tubers or potato plants for the distinctive warty growths on eyes, sprouts, and stem bases. Growths in eyes may be confused with deformations caused by application of sprout retardant (M. C. Hampson, pers. comm.).

2. From plant or nonplant material, collect soil or surface dust for later examination for resting spores. In the field, resting spores have been found in the soil as deep as 50 cm (European and Mediterranean Plant Protection Organization 1980).

Submit for identification, suspect plant material and soil dried and labeled in double containers (one container inside another) with screw tops.

Biology and Etiology

The resting or overwintering spore germinates when spring temperatures rise above 8 °C and soil moisture is sufficient. The spore produces a vesicle that differentiates and releases a sporangium. The sporangium then discharges 200-300 uninucleate, flagellate zoospores, which move through soil water and penetrate a meristematic epidermal host cell (European and Mediterranean Plant Protection Organization 1980, Hampson 1981, Hampson 1986, Sharma and Cammack 1976). The host cell greatly enlarges around the fungus while the fungus develops into 1-9 sporangia (Hampson 1981).

After 36 hours or less, each sporangium discharges 200-300 zoospores (Curtis 1921). The zoospores infect epidermal cells of susceptible tissue about 2 hours after formation (Hampson 1981). Host cells surrounding infected cells enlarge (Curtis 1921) and tissue proliferates, producing the cauliflowerlike warts (Noble and Glynne 1970). The zoospores in turn produce more sporangia, repeating the cycle. Many generations are thus completed during spring or early summer.

In late summer and autumn, two zoospores may fuse to form a diploid biflagellate zygote. The zygote infects a meristematic host cell and develops into a resting spore (Glynne 1925). Surrounding host cells repeatedly divide forming warts (Curtis 1921). The wart quickly decays and releases resting spores into the soil (Hampson and Proudfoot 1974).

Resting spores can germinate after a lapse of a few weeks (Glynne 1925) or several years. Viability in soil lasts for many years and still longer under sod (Pratt 1976); some of the longest reported periods were 25 or more years, 31 years (McDonnell and Kavanagh 1980), and 40 years or more (Laidlaw 1985). These factors combined with a large spore population ensures that infections can occur for many years. Hampson (1979a) estimated that the resting spore population from one infected plant after a natural decline in 22 years from 5 million to 10,000 viable spores, still provided ample inoculum for infection. An inoculum level as low as 60-90 resting spores (European race 2) can cause infection (Hampson and Proudfoot 1974). About 231 and 1,155 resting spores per gram of soil resulted in 10 percent and 70 percent infection, respectively (Glynne 1925).

Susceptible host tissue includes sprouts, stolon buds, stem bases, and young eyes. Infection peaks coincide with production of this tissue. Increase in meristematic tissue due to infection provides additional sites for infection. Until a sprout becomes suberized or corky, it is susceptible to infection (Hampson 1979a, 1981, Weiss 1925).

Soils that are well aerated, tilled, and acid with little potato rotation result in greater disease incidence, but soil type and pH do not appear to be major factors affecting infection. The disease occurs in heavy, medium, and light soils (Bojnansky 1960b) with pH 3.9-8.5, mostly 6-6.5 (Weiss 1925).

Temperature and moisture are the most critical factors determining disease occurrence. Potato wart generally occurs in cool, rainy areas. These and other factors in Europe were discussed by Bojnansky (1960a).

Temperatures during resting spore germination through summer zoospore development ranged 3.5-30 °C. Infections by summer zoospores ranged - 0.8 to 30 °C (Bojnansky 1960a) with optimums of 15-20 °C (Weiss 1925). Of various nearly constant soil temperatures tested, 12-24 °C favored infection; the optimum was about 17-18 °C (Hampson 1981, Hampson and Proudfoot 1974, Hartman 1955, Weiss 1925).

Free water is required for resting spores and sporangia to germinate and for zoospores to disperse (Hampson 1981, Weiss 1925). High soil moisture, however, is less important than periodic watering and drainage of the soil (Weiss 1925), which provides aeration for germination (Bojnansky 1960b). A soil too dry to grow potatoes well was still able to support infection if the soil surface was periodically watered or irrigated (Bojnansky 1968, Weiss 1925).

In Europe, this fungus is favored by cool summers of less than 18 °C, winters of 160 or more days below 5 °C, and annual precipitation of more than 70-80 cm (mostly during summer). In maritime regions, the fungus also thrives where summers are warm (up to a July isotherm of 20 °C) but precipitation is high, or where precipitation is low but summers are colder (Bojnansky 1960a).

In North America, climatic conditions in Newfoundland, Canada, are comparable with Bojnansky's minimum values. There, 25-year averages were July temperatures of 15.6 °C, 181 days below 5 °C, and annual precipitation of about 160 cm (M. C. Hampson, pers. comm).

In the United States, Pennsylvania infestations were located in 120-130 day growing season zones with seasonal mean soil temperatures of about 18-23 °C and 11.4-18.5 cm rainfall monthly. Similar temperatures and precipitation occur in most of the important U.S. potato areas (Weiss 1925).

This fungus was reported once as a vector of potato virus X (Nienhaus and Stille 1965). Transmission has not been confirmed (Lange 1978).

Controls

Strict quarantine measures and restricted planting of susceptible potato cultivars have successfully limited the spread of S. endobioticum. The risk of introduction remains high, however, because of increased international travel, increased movement of potatoes and planting material, expansion of potato breeding programs that may have used wart-susceptible lines, and the existence of many pathotypes (Noble and Glynne 1970).

Planting with resistant cultivars is the only practical means of control. Rotation with nonhosts is ineffective although continuous cropping with susceptible cultivars allows the persistent inoculum to build up in the soil, leading to increased infestations (Ireland Department of Agriculture and Fisheries 1968).

The resting spores are very resistant to fungicides. Effective chemicals in the field are too toxic--killing plants or soil microflora (Walker 1983). While resistant to dry heat, resting spores may be killed by moist heat. Tests with steam at 100 °C for 2.5 minutes (Weiss and Brierley 1928) or immersion in water at 90 °C for 5 minutes (Glynne 1926) killed the spores. Zakopal (1970) studied ways of inactivating the spores in contaminated sewage from processing plants.

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